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INTERMITTENT CLAUDICATION OF THE HIP

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INTERMITTENT claudication has long been recognized as one of the earliest and most frequently encountered symptoms of peripheral vascular disease. It is the purpose of this report to stress the importance of this disease as a cause of localized, intermittent, hip pain.

A cramp-like pain in the calf which occurs during walking and is promptly relieved by rest is almost certain to be correctly attributed to some interference with the normal arterial circulation of the lower leg. Because of the frequent association between pain in the calf and occlusive arterial disease, the term "intermittent claudication" has become almost synonymous with this particular symptom; however, typical intermittent claudication may occur in any muscle or group of muscles if the arterial blood supply is insufficient to permit their normal active use. It most frequently involves the calf for several reasons. These muscles have a relatively large volume yet receive their blood supply through arteries of relatively small caliber. Involvement of these arteries, especially the sural and peroneal arteries, is extremely common in peripheral vascular disease The gastrocnemius and soleus muscles (triceps surae) are the chief plantar flexors of the foot at the ankle joint and are constantly used in walking. When the arterial blood supply to the triceps surae is insufficient to permit the muscle group to continue the vigorous contractions required by a normal amount of walking, running or climbing, claudication develops and is relieved only by cessation of movement.

Intermittent claudication at a level higher than the calf is not a new clinical syndrome. Numerous authors,[1-9] in discussing thrombosis and embolism of the aorta and iliac arteries, have mentioned that their patients sometimes complained of pain in the muscles of the lower back, hips or thighs. Homans[10] in 1939 mentioned the extension of the pain of peripheral vascular disease into the upper thigh and buttocks simulating a sciatic neuritis. In discussing the syndrome of intermittent claudication, Samuels[11] stated that there might be a shooting pain in the buttock of the affected side occurring as a solitary symptom or accompanied by claudication in the legs or feet.

Arteriosclerosis obliterans and thromboangiitis obliterans are the two most common causes of intermittent claudication, although any other condition, local or systemic, which significantly reduces the arterial blood supply to a group of muscles may produce this symptom.

Obliterative arterial disease is seldom considered in the etiology of localized pain in the hip; however, it is an important cause of this symptom. The status of the peripheral circulation in the lower extremities should be ascertained in every patient who complains of pain in the low back, hip or thigh. For example, if a patient is found to have definite impairment of circulation in the right leg and only complains of pain in the right hip, it is possible that the vascular insufficiency alone may be the cause of the pain. This is further substantiated if there is evidence of a better circulation in the opposite limb, and the patient has no complaints referable to this hip or leg. A careful history will enable the physician to decide whether or not the patient has true intermittent claudication of the hip.

During the past five years we have seen 47 patients with occlusive arterial disease involving the abdominal aorta or iliac arteries. The extent of the disease was demonstrated by aortography in 30 of these patients; 26 had typical intermittent claudication involving the hip, thigh or low back area without pain in the calf region.

The following two cases are typical of those studied in this series.

CASE REPORTS

Case 1. A 49 year old businessman was seen at the Clinic on July 27, 1949, complaining of aching in the hips and low back that began after he walked two blocks and that was relieved by rest.

Physical examination was noncontributory. The lower extremities had good pulsations at all levels and there were no physical signs of arterial insufficiency.

X-ray of the lumbosacral spine showed calcifications believed to be in the abdominal aorta and the pelvic vessels. There were hypertrophic bone changes. The examiner believed that the patient's symptoms were due to degenerative joint disease.

Two years later his symptoms had become slightly more severe and he was re-examined. He said that after he walked one block the pain started in his hips and buttocks and progressed into his legs and feet. His feet were always cold and occasionally became numb following exercise. There was no impotency.

Examination revealed the temperature and nutrition of the feet to be good. There were moderate pallor on elevation and rubor on dependency. The venous filling time was 30 seconds bilaterally; this is delayed time consistent with peripheral arterial insufficiency. All pulses were absent except an extremely weak right femoral pulse. Oscillometric readings showed only faint pulsation in the thighs. X-rays again revealed aortic and pelvic calcifications. An aortogram (fig. 1) showed complete obstruction of the aorta just below the level of the renal arteries. The origin of the left renal artery appeared to be slightly narrowed.

He was placed on conservative medical management and has not returned for further treatment.

Case 2. A 62 year old man, a laborer, was first seen at the Clinic on January 29,

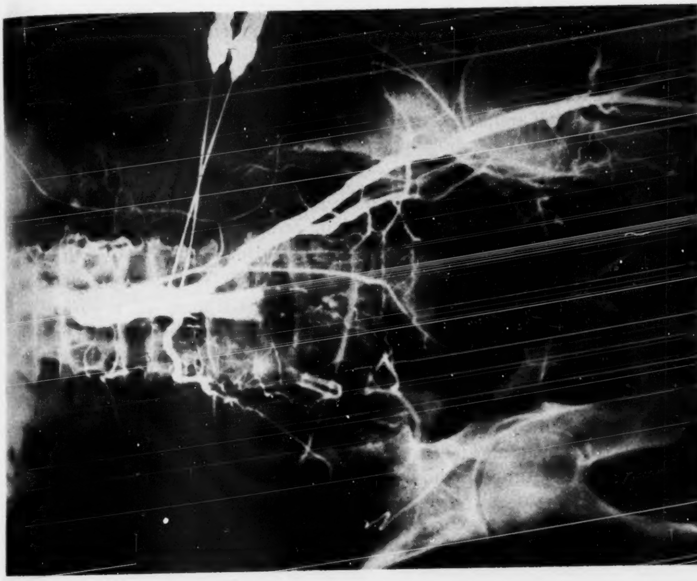

Fig. 2. (Case 2) Aortogram showing occlusion of right common iliac artery at origin. Excellent collateral circulation is demonstrated.

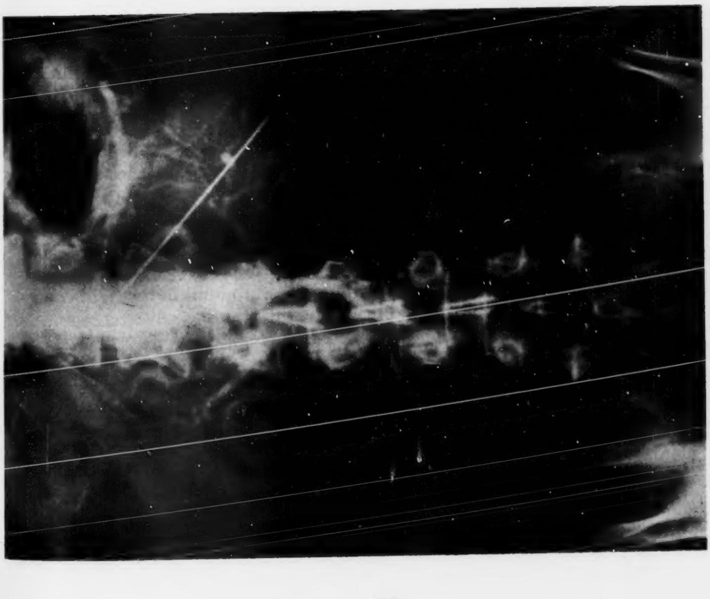

Fig. 1. (Case 1) Aortogram showing complete obstruction of aorta below level of renal arteries.

1952, with a complaint of pain in the right hip and buttock. It had first occurred three months before while he was walking. It had begun suddenly, forcing him to rest, whereupon it subsided in two minutes. Following this, the same discomfort was occasionally incited by prolonged standing or lying on his left side. Then, in addition to the hip pain, he began to experience a weakness in the leg and a numbness down the posterior aspect of the thigh and calf and lateral aspect of the foot. Coughing, straining or motions of the back did not aggravate his discomfort.

After roentgenographic examination, he was told he had osteoarthritis of the spine.

In December of 1952, approximately one year after the initial examination, the patient was seen by a neurosurgeon who noted slight atrophy of the right gluteus muscle and a diminished right ankle jerk. A diagnosis of osteoarthritis of the spine with nerve root compression at S_1 due to a spur or a possible ruptured intervertebral disk was made.

The patient was hospitalized for further study. The spinal fluid was negative. Two diskograms were obtained in December and January and these showed degenerated disks at L_4-L_5 and L_5-S_1, but no protrusion. The pain was not reproduced. A myelogram was negative.

There was no history of diabetes or heart disease.

Physical examination showed a well-developed and well-nourished man. Blood pressure was 190/100 which later fell to normal. A complete examination was noncontributory except for the findings in the lower extremities. There was good nutrition of both feet which were warm and of healthy color. On elevation of the extremities a moderate pallor of the right foot occurred and on dependency a slight rubor. The venous filling time was normal. The peripheral pulses on the left were present and full. On the right only a faint femoral pulsation was felt. Oscillometric examination was normal on the left but abnormal on the right, showing absence of pulsation except for a faint deflection in the thigh.

Laboratory studies including red and white blood counts, hemoglobin, blood sugar and serology were negative. X-rays of the chest and pelvis appeared normal. There was no evidence of calcification of the pelvic arteries. An electrocardiogram was not obtained.

An aortogram (fig. 2) obtained on January 29, 1953, showed occlusion of the right common iliac artery at its origin. There was excellent collateral circulation through the fourth lumbar artery with refilling of the common iliac artery below the block just above the level of the bifurcation into external and internal iliac arteries. There were moderate narrowing of the external iliac artery and slight irregularity of the terminal aorta. The visualization on the left was normal except for some irregularity of the common iliac artery.

On January 30, 1953, one year after the initial examination, a bilateral lumbar sympathectomy from L_1 to L_4 and endarterectomy of the thrombus in the right common iliac artery were done. The patient made an uneventful postoperative recovery.

Approximately two months postoperatively the patient stated that he no longer had pain on walking but had some fatigue in the right thigh after one block of walking, but this was improving. He estimated his improvement to be 75 per cent.

DIAGNOSIS

Intermittent claudication of the hip is not a diagnosis in itself, but rather a symptom of peripheral vascular disease. Arteriosclerosis obliterans is the most common cause of this symptom. Either the abdominal aorta or the iliac arteries

or both may be involved. Occasionally, roentgenograms of the pelvis will reveal more or less extensive calcification in these vessels, but the simple presence of this calcification does not necessarily indicate the presence of claudication. Extensive calcification of the Mönckeberg's type does not usually give rise to symptoms of peripheral vascular disease. The extent of the occlusive arterial disease was demonstrated by aortography in 30 patients in this series. This is a safe procedure and has been invaluable as a diagnostic aid in this group.

Various orthopedic and neurosurgical conditions must be considered in the differential diagnosis of intermittent claudication of the hip. These diseases may be readily differentiated through evaluation of the status of the circulation in the lower extremities of those patients who complain of pain in the low back, thigh and leg.

Hypertrophic arthritis of the hip (malum coxae senilis) is an extremely common cause of pain in the hips of patients more than 50 years of age. These patients complain of pains in the muscles about their hips which are aggravated by activity and relieved by rest. However, some restriction of movement of the hip joint always accompanies this pain and roentgenograms reveal the typical cystic and sclerotic changes in the acetabulum and the head of the femur. Although there may be evidence of some arteriosclerosis of the vessels of the involved extremity, there is usually none of any severe impairment of the peripheral circulation.

A localized fibrositis or a simple nonsuppurative bursitis is the most common cause of pain in the hips of patients younger than 50 years. Eighteen or more different bursae have been described about the hip joint, but of these only four are of clinical importance: iliopectineal, ischiogluteal, superficial trochanteric and deep trochanteric. Patients with bursitis have extreme tenderness immediately over or adjacent to the bursa and passive movement of the hip joint usually aggravates the pain. Frequently, those with fibrositis have "trigger points" or areas of extreme tenderness either in the muscle bellies or at the tendinous attachments of muscles to bone. Local infiltration of these tender areas with a dilute solution of novocain will usually give immediate relief from pain.

A protruded intervertebral disk in the lower lumbar region may simulate a true intermittent claudication of the hip. Two of the patients in this series were first diagnosed as having a protruded intervertebral disk and were referred for surgical treatment. It is true that any pressure or irritation of the fifth lumbar or first sacral nerve root may produce pain in the posterior hip area, radiating down the leg in the course of the sciatic nerve. The pain due to a protruded disk is almost always aggravated by bending, lifting, coughing or straining. In contradistinction to intermittent claudication of the hip, the pain associated with a protruded disk is not necessarily dependent upon movement for its production or aggravation. A protruded disk usually produces some alteration in the deep tendon reflexes in the involved lower extremity; and there may also be characteristic sensory changes and muscular weakness in the leg.

Any of the malformations and diseases involving the structures about the

hip may be accompanied by some intermittent pain in this area. The restriction of passive movement in the hip by muscle spasm, bony abnormality or incomplete fibrous ankylosis and the presence of an adequate peripheral circulation in the lower extremity readily differentiate these conditions from intermittent claudication of the hip.

SUMMARY

Pain in the low back, hip and thigh may be of vascular origin due to occlusive arterial disease involving the abdominal aorta or iliac arteries. Intermittent claudication of the hip may be mistaken for various orthopedic and neurosurgical diseases. A careful history of the character and duration of the pain and a careful examination of the peripheral circulation will always differentiate true intermittent claudication of the hip from any other disorder producing low back pain, hip pain or sciatica. Aortography is a valuable diagnostic aid.

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PENTOTHAL ANESTHESIA IN INFANTS AND CHILDREN

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MANY anesthesiologists consider intravenous pentothal sodium anesthesia to be contraindicated in children. Antipathy towards the agent arose with the early practice of using it in large doses and high concentrations. Since the limits of physiologic balance in infants and children during anesthesia are so narrow, only small deviations from the normal can be tolerated for any length of time.[1] The overwhelming quantities of pentothal which were introduced directly into the circulation produced prompt respiratory and subsequent circulatory collapse. A few catastrophies tended to discourage further clinical trial.

When small calculated amounts of pentothal are used in conjunction with a few precautionary measures, this agent not only affords safe anesthesia[2] but offers a technic which decreases operating time by permitting electrocoagulation and allows the anesthetist to be remotely placed from the patient. These factors are particularly advantageous during neurosurgical procedures and surgery of the head and neck.

METHOD

The infant or child is intubated under ether anesthesia by the open drop method. In the average child a combination of morphine and atropine sulfate according to age and weight is sufficient premedication. In the excitable child, supplementary agents such as pentobarbital or rectal pentothal may be required for sedation to avoid the psychic trauma of ether induction. To force an anesthesia mask upon the face of a terrified child is cruel, especially when such action can be easily avoided by adequate premedication.

In early third plane of surgical anesthesia, an oro-tracheal tube is inserted into the trachea at least 2 cm. beyond the larynx. The tube should be carefully selected because, if too large, it produces postoperative laryngeal edema, and if too small, it deprives the anesthetist of adequate control of the respiratory system by leakage and prevents use of positive pressure breathing.

The small larynx of the newborn child will accept an 18 fr. endotracheal catheter. For older children, a larger size is selected which approaches the size of the laryngeal introitus but does not produce unnecessary pressure upon the vocal cords. For a child one year of age a 20 fr. endotracheal tube is usually selected and for a child six years of age a 24 fr.

The trachea of a newborn infant is 4 cm. in length[3] (Tables 1 and 2). By inserting an endotracheal catheter 2 cm., the tip will lie midway between the

Table 1

Length of Trachea in Relation to Age of Patient

Age	Length in cm.
Birth	4.
1-2 yrs.	4.5
6-8 yrs.	5.7
14-16 yrs.	7.2
More than 16 yrs.	9-15

Table 2

Sagittal and Coronal Diameters of the Trachea in Relation to Age of Patient

Age	A-P diameter in mm.	Transverse diameter in mm.
Birth	5.7	6.
1-2 yrs.	9.4	8.8
6-8 yrs.	10.4	11.0
14-16 yrs.	10.7	13.5
More than 16 yrs.	16.5	14.4

larynx and the carina; the tip should never be inserted to or below the level of the carina. In most age groups, intubating the larynx 2 cm. is adequate. When the catheter is anchored satisfactorily, accidental extubation is rare and endobronchial intubation extremely unlikely.

Endobronchial catheterization causes insufficient aeration of the opposite lung, resulting in carbon dioxide retention. The usual signs of hypercarbia (e.g. hyperpnea) may be masked by the depression due to pentothal. The increase in respiratory rate may be interpreted as a sign of lightening of anesthesia and mistakenly controlled by giving additional pentothal. To anchor the endotracheal tube in its proper place and to prevent its slipping into a main stem bronchus, it is fixed at the lips by adhesive tape which binds the tube to the gauze bite block and extends across both sides of the face to the cap (fig. 1).

Pentothal is administered intravenously. A venotomy is performed under ether anesthesia on the saphenous vein at the anterior aspect of the internal malleolus (fig. 2). The vein is exposed and isolated from all surrounding structures. Two ligatures are placed around the vessel. Traction is made on the distal one, and a three-cornered incision is made in the lumen of the vein. A short length of plastic tubing (polyethylene) is threaded into the lumen for a distance of 7 to 10 cm. and secured by tying the uppermost ligature. The free end of the polyethylene tubing is then adapted to the disposable venoclysis set (dextrose 5 per cent in water—250 cc.) by inserting the proper sized needle into the intravenous polyethylene tubing (fig. 3). If blood is to be transfused, the larger intravenous tubing (No. 190) is preferred. For polyethylene tubing

No. 90, a 20 gauge needle is used and for No. 190, an 18 gauge needle. Tying the lower ligature prevents bleeding from the lower segment of the vein. To secure the intravenous polyethylene tubing adequately, the adapting needle hub is sutured to the skin (fig. 3). When the skin incision is closed, a gauze sponge is placed over the incision and the whole anchored with adhesive tape or roller bandage, and the venoclysis started at an extremely slow rate (20 to 30 drops per minute).

Pentothal is administered by the system illustrated in Figure 3. A 20 gauge needle is inserted through the latex rubber portion of the venoclysis tubing and the needle point advanced into the nylon tip. This needle is connected to the small white rubber tubing leading to the pentothal syringes. A series of clamps permits the infusion of finely graduated doses of a 1 per cent solution of pentothal sodium. The small (2 cc.) syringe is graduated in 1/10 of 1 cc., and contains a total of 40 mg. of pentothal. The large (30 cc.) syringe is filled with the pentothal solution and serves as a reservoir.

An injection of 10 mg. of pentothal is the initial trial dose, allowing the anesthetist to judge the individual tolerance of the patient. Since the pentothal enters the venoclysis system so close to the vein, only a few centimeters of the dextrose solution is necessary to carry it into the circulation.

The level of anesthesia under pentothal is judged by the rate and depth of

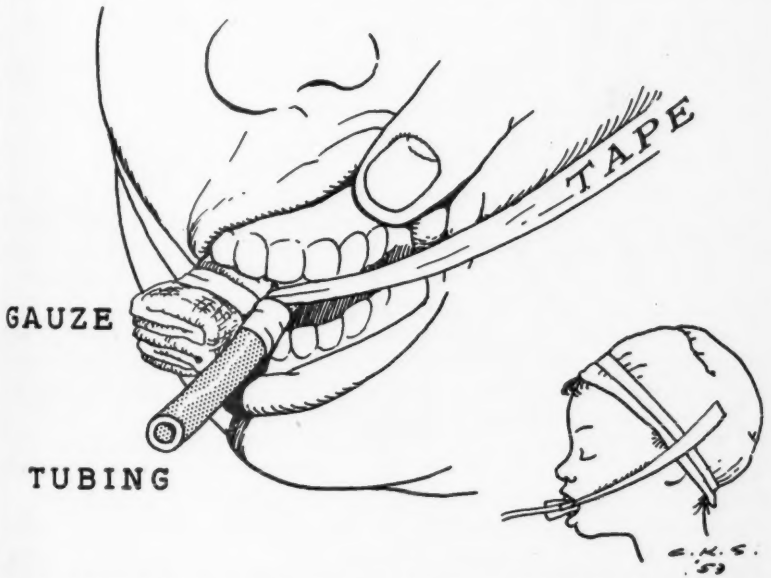

Fig. 1. Endotracheal tube is anchored at the lips. Gauze bite block is placed between the teeth to prevent biting tube and obstruction of the lumen.

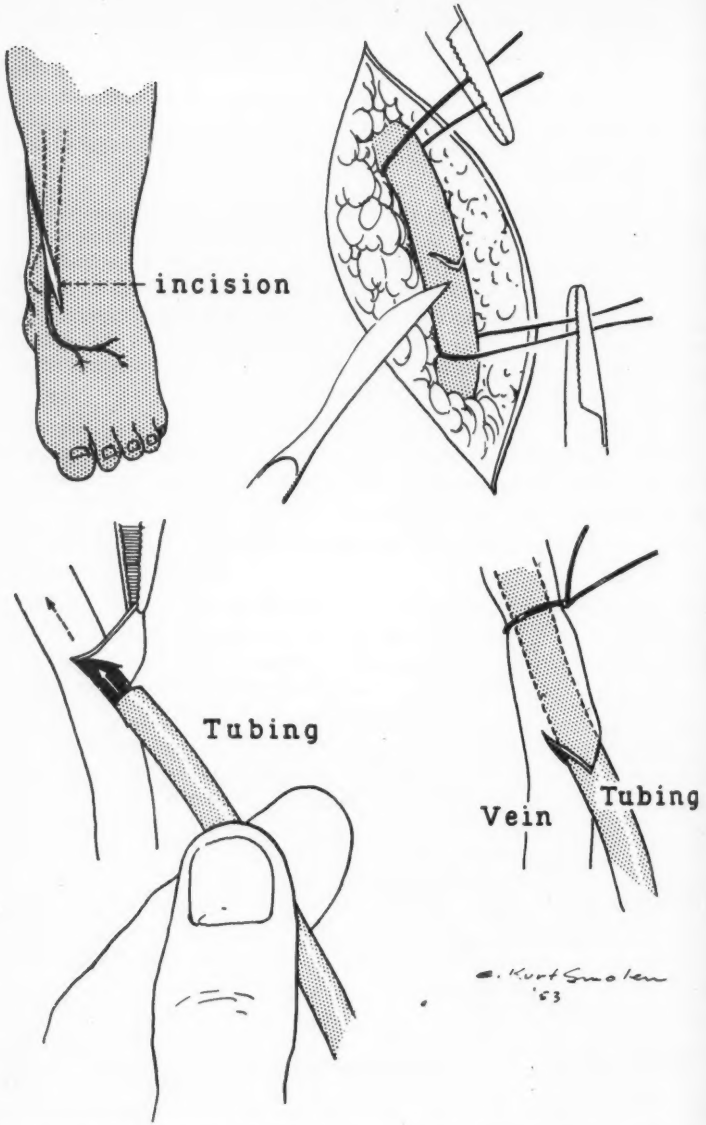

Fig. 2. The site of incision for cut-down on the saphenous vein at anterior aspect of the internal malleolus is shown. A three-cornered incision is made in the isolated segment of vein and a short length of polyethylene tubing inserted. A blunted needle adapts the venoclysis tubing to the polyethylene tubing.

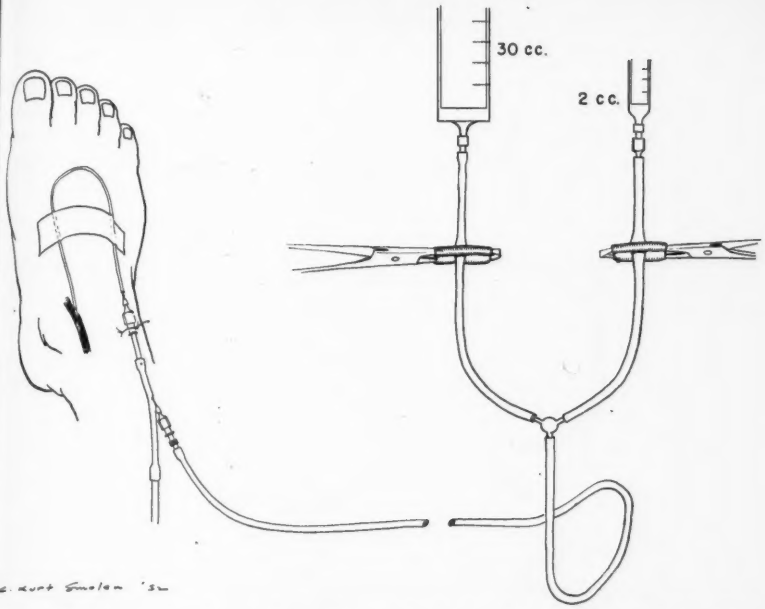

Fig. 3. The set-up for the administration of graduated dosages of pentothal includes a 30 cc. syringe or reservoir and 2 cc. syringe. The tubing is connected to the venoclysis set close to its entrance into the vein. An injection of pentothal then enters the circulation immediately.

respirations. Increasing rate denotes a light plane and too great a dosage causes apnea. Surgical anesthesia occurs when the respirations are slow (18 to 22 per minute) and of average depth. Once a desired level of hypnosis is acquired, maintenance doses are minimal and their effect more prolonged.

If apnea ensues, anoxia is prevented by artificial respiration. By means of the TO & FRO system (fig. 4), manual control of the respiratory system is maintained. This closed system allows the anesthetist to inflate the lung forcibly by positive pressure breathing. Should the contemplated surgical procedure necessitate the prone position, respiratory assistance must be given by aiding each respiration. Only in this way can adequate oxygenation and carbon dioxide exchange be guaranteed.

The endotracheal tube is removed as soon as the patient has reacted and while he is still in the recovery room. Early extubation may prevent laryngeal edema by eliminating further irritation of the mucous membrane of the trachea and larynx. Oxygen is made available by using a funnel directly in front of the infant's face. In most instances it is impossible to utilize a nasal catheter for oxygen.

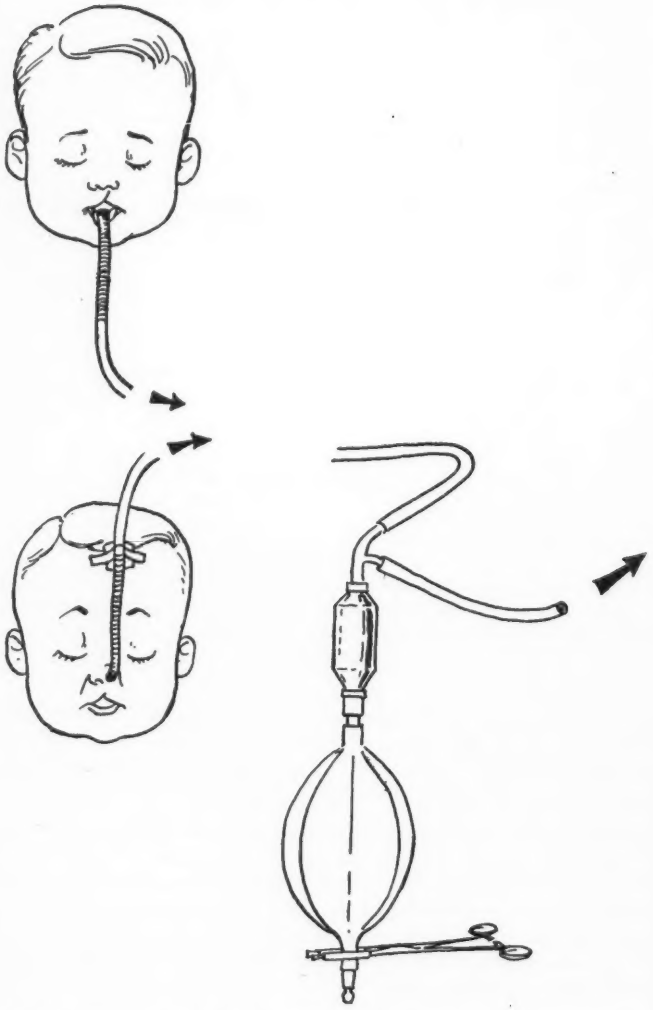

Fig. 4. A new longer endotracheal tube fits the contour of the face and extends out of the surgical field. No adapters and angle connectors are necessary. The distal end of the various sized tubes are all the same size and adapt snugly to the standard equipment.

COMMENT

Intravenous pentothal anesthesia in children is especially adaptable to surgery of the brain, head and neck. This method has been used in 133 neurosurgical cases at the Cleveland Clinic in the past two years (1951 to 1953) without serious anesthetic complications or death. The age distribution of these patients is presented in Table 3. The youngest patient was eight days old.

Table 3

Distribution According to Age Groups of Neurosurgical Patients Given Pentothal Anesthesia

(1951-1953)

Age Range	Incidence
Birth to 3 mo.	12
3 mo. to 1 yr.	41
1 yr. to 3 yrs.	33
3 yrs. to 6 yrs.	18
6 yrs. to 10 yrs.	29
TOTAL	133

With this type of anesthesia the anesthetist can be placed in a more remote position, allowing the surgeons more room and better exposure. Since explosive agents are not used, cautery and electrocoagulation may be employed freely without danger. When supplemented with local procaine infiltration for incision, light pentothal is used only to keep the child asleep and prevent movements. This results in less shock and rapid postoperative recovery. On the other hand if large amounts of pentothal are used indiscriminately, the postoperative recovery will be extended and the patient may not react for hours.

It has been found advantageous to use a tube longer than that of the usual endotracheal system, so that the head and face are not cluttered with anesthesia equipment. However, the respiratory dead space must not be increased beyond physiologic limits. The patient is under good control, the numerous metal endotracheal adapters are eliminated and the tube may be made to fit the contour of the face. The catheter is connected to the standard end of the TO & FRO canister. A spiral nylon filament supports the walls of the latex rubber catheter and prevents obstruction of the lumen when the catheter is acutely bent.

SUMMARY

1. Intravenous pentothal anesthesia is a safe and easily adaptable anesthesia for infants and children.

2. Oro-tracheal or nasotracheal intubation is necessary for safe pentothal anesthesia in infants and children.

3. A venous cut-down technic utilizing the polyethylene catheters is described for administration of venoclysis and pentothal.

4. An endotracheal tube, longer than the usual catheter, eliminates the numerous metal angles and adapters and allows the patient's head and face to be free of anesthesia equipment.

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FACTORS IN THE MECHANISM OF METASTASIS: A REVIEW

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THE phenomena displayed by malignant neoplasms of invading locally and disseminating widely have been observed almost since cancer has been recognized. The routes by which these tumors metastasize to distant sites have been frequently discussed in the medical literature. Also the more or less definite patterns of metastatic lesions in many malignant tumors have been observed and recorded. However, the mechanisms involved in the dissemination of tumors have only recently been studied by modern biochemical, microdissection, tissue culture and histologic technics. It is not possible to consider each type of malignant neoplasm separately, but to our knowledge the fundamental mechanisms to be discussed in this article are applicable to malignant tumors in general.

LOCAL INVASION

There is almost no malignant tumor in which local invasion is not observed to a greater or a lesser extent. In many this property is well developed and in others the local infiltration of tissues is desultory. Coman et el. and others[1-5] in considering this problem proposed that invasive growth depends upon three factors: (1) decreased adhesiveness of cancer cells; (2) ameboid movement; and (3) liberation of a spreading factor.

(1) **Decreased Adhesiveness of Cancer Cells.** The initial work on the cellular adhesiveness of cancer cells dealt with a comparison between squamous cells from normal lips and those from squamous cell carcinomas of the lip.[1,2] By means of a microdissection technic, the force required to separate cells from one another was gauged and cancer cells were shown to require less than half the force necessary to separate normal ones. During the process of separation normal cells showed distinct tension lines, became elongate, detached with a snap, and finally resumed their normal shape. In malignant squamous cells there was much less distortion and the separation was readily accomplished. Further experiments employing an agitating technic subjected samples of normal and neoplastic cells to varying degrees of violent shaking. The exfoliated single recognizable cells were then counted and in all cases of malignant tumors the cell counts were considerably higher. These experiments have included carcinomas of hollow viscera, cervix and breast. In all of these the results were constant, indicating that decreased adhesiveness is a property of cancer cells.

What is the reason for this decreased adhesiveness of cancer cells? Is it some

alteration in the chemical make-up of the cells? Coman, Brunschwig and others have shown that a decreased calcium content in cancers is a constant finding.[4, 6-8] By means of direct calcium determinations and flame spectophotometer examinations the calcium content of tumors and of normal adjacent tissue were compared. Potassium determinations were also made. In carcinomas of the colon the calcium content averaged 44 per cent less than the normal tissues and the potassium averaged 60 per cent higher. The decreased calcium content was interpreted by Coman to be an expression of invasiveness of cancer cells and a factor in their decreased adhesiveness. The elevated potassium content is more definitely understood. Rat experiments on regenerating liver tissue showed that potassium was elevated in tissues taken from actively growing zones in comparison with that from surrounding normal tissues. This elevated potassium was correlated with the numbers of mitoses present and was found to be directly proportional to the rate of mitosis.[8] Similar observations have been made on human tissues. Therefore, elevated potassium levels are an expression of cellular multiplication.

(2) **Ameboid Movement.** Active ameboid motility has been observed in cultures of both animal and human neoplastic tissue.[9] Many different types of tissues in tissue culture show cells which detach themselves from the central portion of the culture and exhibit ameboid locomotion. In studies on breast cancers this motility was appreciable and averaged 0.7 micra per minute.[9] Renal cell carcinomas, oncocytomas, and leiomyosarcomas all showed similar motility. However, as far as can be determined, ameboid activity of tumor cells in vivo has not been demonstrated although it may well occur. If it does, it would aid in the explanation of the behavior of malignant tumors both as to local invasion and as to the production of metastases.

(3) **Liberation of a Spreading Factor.** The presence of a spreading factor similar to hyaluronidase has been demonstrated in some tumors.[3,10] This substance hydrolyzes the hyaluronic acid of the intercellular cement substance of connective tissue and permits the penetration by malignant cells. In view of the fact that only some tumors produce hyaluronidase or a spreading factor, it cannot be regarded as a requisite for local invasiveness of cancer cells, but neither can it be disputed that the presence of such a factor would greatly facilitate the process of local invasion.

METASTASIS TO DISTANT SITES

The routes by which malignant neoplasms metastasize are well known and may be grouped under three headings: lymphatic, hematogenous and transcoelomic. It is axiomatic that tumor emboli gain access to lymphatics, to blood channels, or are cast free in any of the serous cavities by means of local invasion. However, certain purely physical factors aid emboli in gaining access to these structures. Studies by Young and Griffith[11] indicated that all tissue including neoplastic exemplify a differential pressure system in that they consist of vascular tubes invested by a semi-solid medium. Experiments showed that

emboli or other bodies cannot enter collapsible tubes or lymphatic or blood channels as long as the pressure within the channel is greater than the pressure surrounding it. Thus anything which increases the tissue or surrounding pressure favors embolization. The erosion and subsequent rupture of a vascular channel within a tumor may produce such circumstances. Active growth of a neoplasm confined to a more or less limited space will increase tissue pressure and provide the added physical factor. Manometric studies have confirmed this and have also shown that injection of saline or other fluid and digital compression increase tissue pressure.[12] From these facts it can be concluded that a malignant tumor in itself provides the locally invasive cancer cell or embolus and in many instances the required increased tissue pressure by its own growth. The unfortunate addition of further mechanical factors can only serve to augment an already undesirable set of circumstances.

Thus far we have considered local invasion and the mechanical factors in metastasis. What are the influences governing the number of metastases?

Many if not most tumor emboli fail to survive the embolism. Zeidman, McCutcheon, Coman,[13] and others have studied this problem extensively using transplantable mouse tumor. Determined concentrations of cells of mouse sarcoma 241 were prepared, injected intravenously into C57 mice, and 18 days later the resultant metastases counted. Relatively large numbers of tumor cells were required indicating that many of the tumor cells failed to survive the embolism. But in humans, emboli are given off probably at a sporadic but more or less constant, slow rate quite dissimilar to an injection. Therefore, a corollary experiment was performed in which large and small implants of the same tumor were placed in the flanks of mice and the animals killed at intervals. The results showed that the duration of the primary was a determinant in the number of metastases and that greater numbers of metastases resulted from large than from small implants. However, there was no definite correlation between the final size of the primary and the number of metastases.

It is a common observation that thyroid, spleen, and skeletal muscle are seldom the seat of metastases. Coman[14] pointed out, though these structures have often been considered as offering "poor soil," experimentally this is not true since intra-arterial injections of a suspension of V_2 rabbit carcinoma into rabbits produced neoplasms in skeletal muscle as well as in any other location in the body. Injections into the left side of the heart produced metastases in myocardium, skin, skeletal muscle, lungs, kidneys, liver and other viscera. In this instance metastases were frequent in the muscles of the trunk and became less frequent in the distal portions of the extremities. This tumor can grow in all tissues of the body, yet of itself the neoplasm rarely metastasized beyond the regional nodes and the lungs. Injections of the tumor in the femoral vein produced no tumors beyond the lungs indicating that some of the cells failed to survive the embolism and the remainder were arrested in the lungs.

The question then arises: How effective are the lungs in filtering out tumor cells and do cancer cells pass through the pulmonary circuit without being arrested? Experiments using three different types of experimental cancers were

employed to solve this problem.[15] Cell suspensions of the V_2 squamous carcinoma, the Brown-Pearce carcinoma, and the Walker rat carcinoma 256 were injected into the veins of one group of rabbits and almost immediately the aortic blood collected. The aortic blood was then injected into a second group of rabbits. Autopsies done on the second group three to five weeks later showed some interesting results. With the Brown-Pearce carcinoma 10 of 20 rabbits developed metastases to distant sites and in five of the ten no pulmonary lesions were found. The V_2 carcinoma and the Walker 256 carcinoma produced lesions in 2 of 15, and 1 of 11 respectively. In view of these results it is inescapable that some tumor cells can pass through the pulmonary circuit and enter the systemic circulation. These experiments are doubly remarkable since only 50 cc. of aortic blood was employed.

Since certain types of experimental tumors passed through the pulmonary circuit more readily than others, the same authors examined these tumors for differences in cell size and found no significant difference. Passage of tumor cells through an arteriovenous shunt or through a patent foramen ovale could be excluded since so few cells of the V_2 carcinoma and the Walker carcinoma were able to pass through the lungs. It can be concluded that some types of tumor cells are better able to effect transpulmonary passage than others. The reason for this difference is still obscure although it is perhaps due to variations in pliability and viability of tumor cells.

In order to explain the reasons for the rarity of secondary tumors in some organs, Coman et al.[16] conducted the following experiments. Tumor cells stained with iron hematoxylin were injected into the left side of the heart of rabbits, and the animals killed in one to three minutes following injection. Multiple sections were then taken and examined for the presence of stained cells. The cells were counted and it was noted whether they appeared in capillaries or in arterioles. Emboli were found everywhere, but in the spleen and thyroid they were arrested in arterioles with only a few in capillaries. Emboli were rare in muscle. When viable tumor cells were used and the animals autopsied in one to three weeks, a close correlation between the number of emboli lodging in capillaries and the number of metastases was noted. When emboli in arterioles were included the correlation was lost. In the spleen and thyroid almost all of the emboli were in arterioles and secondary neoplasms not produced. In addition tumors most often developed when emboli of four or less cells were present. It is therefore established that tumors most often develop when emboli of four or less cells are arrested in capillaries and they are rare where emboli are arrested principally by arterioles.

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THE VALUE OF AIR ENCEPHALOGRAPHY AND CEREBRAL ARTERIOGRAPHY IN THE DIAGNOSIS OF HEADACHE

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ALTHOUGH pain in the head is a common and stubborn complaint, the underlying cause usually is benign, and its source more often outside the cranium than within.

The most common pain producing mechanism is an excessive relaxation and dilatation of the extracranial arteries; these vessels are for the most part branches of the external carotid artery. Thus, extracranial vascular headache is the most prevalent type. As a consequence of difference in location and clinical pattern, various names have been given to this form of head pain: migraine, atypical migraine, histamine cephalgia, atypical facial neuralgia, tension vascular and hypertensive headache. The underlying mechanism appears to be similar in all of these, and basically they are all related. A strong familial tendency has also been noted, and emotional and nervous tension factors usually play an important role. It frequently occurs in a setting of tension, fatigue, or frustration, or in the period of relaxation afterwards.

The second most common pain producing mechanism is related to sustained contractions of the muscles of the head and neck which give rise to a muscle tension headache. This may be primary or secondary to any other pain in the head. As is true of the extracranial vascular type, the primary muscle tension headache is usually due to various emotional and nervous factors.

In the past the role of the eyes, nose and sinuses in the production of head pain has been stressed. Notwithstanding the voluminous literature on the subject, it appears that these structures do not commonly produce this symptom; at least they are not responsible for the many severe forms constantly reported by patients to the headache consultant.

Although the great majority of chronic and recurring headaches are extracranial in origin and benign, the possibility of an intracranial lesion should be considered. Usually the differentiation can be made without much trouble, but sometimes complicated neurologic studies must be carried out. Many patients seek medical attention, not because the pain is intolerable, but because there is an underlying fear of brain tumor. Such patients must be reassured, and most of them can be reassured without resorting to neurosurgical diagnostic procedures. The indiscriminate use of air encephalography, ventriculography, and cerebral arteriography in patients with benign syndromes is to be condemned. These procedures are not entirely innocuous, and they are expensive. When not indicated, they may do a great deal of harm by creating further

apprehension or serving to fix the headache. The financial burden alone may be enough to create further headache.

Yet, the air encephalogram and cerebral arteriogram are valuable and necessary diagnostic aids. Positive evidence of an intracranial lesion allows a direct surgical attack with the resultant saving of lives; evidence ruling out an intracranial lesion may be equally valuable. Since headache is the first symptom of one third of supra-tentorial tumors and is usually the first symptom in posterior fossa tumors, except for those located in the cerebellopontine angle,[1] the advisability of obtaining an air encephalogram or arteriogram is frequently a problem. If there are positive physical or x-ray findings, the decision is relatively easy. Abnormality of the spinal fluid warrents further study. A well-taken history is often of great help although the severity of the headache alone should not be a determining factor, since the benign variety is frequently the most severe. When headache is the only symptom and the neurologic, roentgenologic, and spinal fluid studies are normal, how often are neurosurgical diagnostic procedures indicated? If carried out, how often is an intracranial lesion demonstrated? In an attempt to answer these questions, the following study was made.

Method

The records of all patients in the Department of Neurological Surgery of the Cleveland Clinic undergoing air encephalography, cerebral arteriography, or both during the years 1948 and 1949 were reviewed. These years were chosen in order to allow sufficient time for adequate follow-up. Selected for study were those patients who complained only of headache and in whom the neurologic examination, x-ray studies and spinal fluid were entirely normal. There were 43 such patients, 21 men and 22 women, during the two year period. Their average age was 35 years. In addition to the previously described procedures, electroencephalograms had been obtained in 16 instances: nine were found to be normal, five possibly abnormal, and two abnormal. Of these 43 patients, 34 were subjected to air encephalography, 4 to cerebral arteriography, and 5 to both procedures.

Result

From a positive standpoint the result of the neurosurgical diagnostic procedures in these cases was most discouraging. All of the air encephalograms and cerebral arteriograms were normal, and not a single organic lesion was demonstrated. In so far as is known, no intracranial lesion appeared subsequently. This does not mean the procedures were totally unprofitable, for the presence of an intracranial lesion could be ruled out in each case. In this regard the tests were for the most part justifiable and in many instances truly necessary. However, it is probable that many of the studies were ordered merely because of the presence of severe recurrent headache. Each case must be evaluated critically. There is still no substitute for a carefully taken history interpreted in the light of wide experience by a physician interested in the problem of headache.

CONCLUSIONS

1. In a series of patients seen during a period of two years with the single neurologic complaint of headache, in whom the neurologic, roentgenologic and spinal fluid examinations were normal, air encephalography, cerebral arteriography, or a combination of these two procedures were carried out in 43 instances.

2. Not a single organic intracranial lesion was demonstrated.

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POLYPS OF THE DIGESTIVE PHARYNX

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POLYPS of the oro- and hypopharynx are of rare occurrence. Only four cases under this classification have been reported since 1920. However, in 1942 Samson and Zelman[1] reviewed the literature and found 25 reports of cases of "pedunculated tumors of the esophagus;" to these they added one of their own. In 15 of the 19 cases in which the area of attachment of the pedicle of the tumor was recorded in their report, it was at or above the level of the cricoid cartilage which anatomically makes them lesions of the hypopharynx. These 15, in addition to the four reported since 1920, bring the total number of recorded cases of polyps of the digestive pharynx to 19. An additional case will be reported here.

CASE REPORT

A 48 year old white man was first seen at the Cleveland Clinic on August 23, 1951, with a one year history of a lump in the left side of the throat which caused him to have a constant desire to clear his throat. Occasionally, he had been able to bring the mass

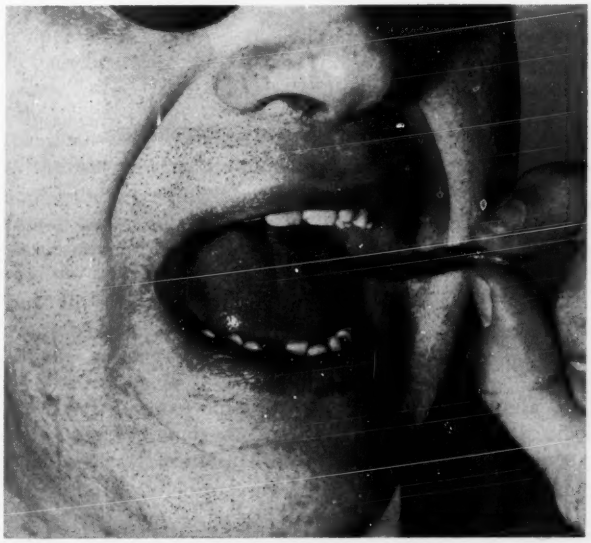

Fig. 1. Photograph of patient showing regurgitated polyp.

up into his mouth where it would protrude beyond his lips. On two occasions he had choked on the lump and had difficulty in getting his breath for a short period.

Examination, with the mass regurgitated into the mouth, revealed a slender pedunculated tumor apparently arising from the left pyriform sinus (fig. 1). After the patient re-swallowed the mass, it could not be visualized by mirror laryngoscopy.

On September 7, 1951, 15 days after the initial examination, the patient was operated upon under pentothal and nitrous oxide anesthesia using a naso-tracheal airway. The hypopharynx was exposed with a David-Crowe mouth gag. The pedicle of the polyp, approximately 1 cm. in diameter, was found to be attached to the left lateral pharyngeal wall at the level of the arytenoid cartilage. The lesion was removed flush with the pharyngeal wall using a tonsil snare. Bleeding was controlled by coagulation diathermy.

The postoperative course was uneventful and the patient was discharged the following day. When last seen on December 24, 1952, approximately 15 months after operation, he had remained symptom-free.

The specimen after fixation measured 4.5 cm. in length and 1 to 1.5 cm. in diameter (fig. 2). The surface was smooth and glistening. One section at the tip revealed a surface

Fig. 2. Operative specimen. Some shrinkage from fixation.

ulceration containing purulent exudate. The bulk of the specimen was formed by hyaline and edematous connective tissue with islands of fat tissue. The surface was covered by well-differentiated squamous epithelium. The pathologic diagnosis was laryngopharyngeal polyp with ulceration and marked chronic inflammation (fig. 3).

COMMENT

In the 20 cases, including those already reported and that presented here, the pathologic diagnoses have been: lipoma, myxoma, fibroma, fibro-epithelial polyp, and benign fibromatous polyp. In two instances the lesions were multiple.

Symptoms reported have included: regurgitation of the polyps, intermittent dysphagia, lump in the throat, throat clearing, cough, dyspnea, hoarseness, wheezing, nausea and vomiting. Three patients have died of asphyxia due to aspiration of polyps.

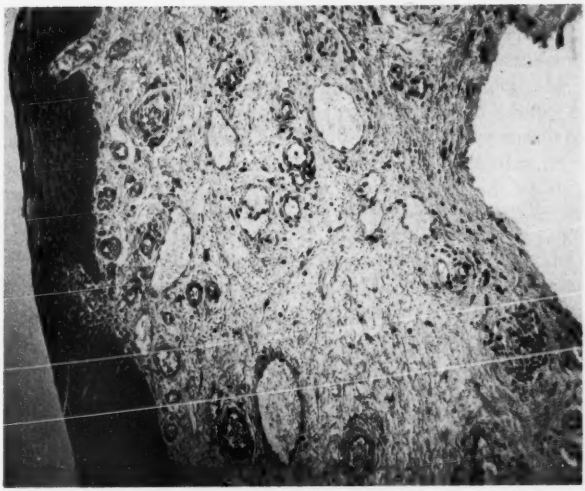

Fig. 3. Laryngopharyngeal polyp. (See text.)

The ages of these 20 patients ranged from 26 to 81 years. Only two were women.

Malignancy in these tumors must always be excluded. In a recent unreported case of an apparently benign polyp arising from the pyriform sinus, sections through the pedicle revealed epidermoid carcinoma. Barium esophagram may or may not show an elongated filling defect of the upper esophagus. Mirror laryngoscopy might easily fail to reveal any abnormality. If the pedicle is not easily visible, such a lesion is sometimes overlooked on esophagoscopy due to blending of the polyp with the normal esophageal mucosa.

Considering the preceding factors and the frequent absence of the symptom of regurgitation of a mass into the mouth, one might wonder how many patients with lesions of this type are diagnosed as having complaints of a functional nature.

Although polyps of the digestive pharynx are rare, as witnessed by the paucity of available reports, they achieve importance because occasionally aspiration of the tumor causes asphyxia which results in the patient's death.

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THE HUMAN SIDE OF SCIENCE

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ALMOST 60 years have passed since Wilhelm Conrad Röntgen, professor of physics at the University of Würzburg (in November 1895), saw the effect of a strange and unusual phenomenon while he was performing some experiments in his laboratory. This was the bright fluorescence of some barium platinocyanide crystals near an electrically excited Hittorf-Crookes tube. He pursued the study of this effect in a most masterly and thorough manner, and discovered it to be due to "new kind of rays," which he called the "x-rays" and which now are often called the "roentgen rays." This famous discovery, which so profoundly influenced many branches of science and medicine, placed Röntgen in the ranks of the world's great men.

With a discovery so epoch-making as that of the x-rays, and in view of the immediate and unprecedented interest in it on the part of the scientific world and the general public, it was perhaps inevitable that some confusions and even unjust criticisms concerning it should arise to disturb and plague Röntgen. Because he reacted to criticism with great sensitivity, and even bitterness—though he also did his best to avoid and evade acclaim—an account of these negative unpleasant accompaniments of the discovery forms an interesting chapter on scientific controversy and polemics.

Some of the controversy regarding priority in regard to certain phases or features of the discovery was undoubtedly not inspired by dishonesty, but rather by the confusion which arose naturally from the tremendous publicity concerning the new scientific wonder and ignorance concerning its real nature. The distinction between the new rays and the well-known cathode rays was confusing to nonscientific minds, and this confusion was evident in some of the stories that were circulated regarding the discovery. Another factor which gave rise to some of the unfounded and inaccurate accounts of the event was the desire of certain little persons in the periphery of Röntgen's environment to gain personal prestige by pretending to know some "inside story" connected with it. These gossipy rumors and descriptions undoubtedly were exaggerated and magnified by Röntgen's aversion to publicity and his own reticence in discussing the actual happenings.

In addition to these factors, it is unpleasant to record that evidently most of the unfair criticism came from certain scientists who were jealous of Röntgen's success. Many prominent scientists had been investigating the cathode rays, and felt chagrin that they had failed to observe the phenomena which led Röntgen to detect the existence of the x-rays, although undoubtedly x-rays had been produced in their experiments, as was recognized after the discovery was announced.

False claims of priority and attempts to belittle the importance of the discovery and Röntgen's genius in making it were already appearing early in 1896, even before the first tumult of acclaim and praise had subsided. Although these criticisms were puzzling and disturbing to Röntgen, he apparently resolved to ignore them philosophically, as he indicated in a letter to his friend Ludwig Zehnder: "My work has received recognition from many quarters . . . This is worth a great deal to me, and I let the envious chatter in peace; I am not concerned about that." Nevertheless, the record and his own behavior later clearly demonstrate that he was concerned about it, and became increasingly so with the passage of the years. He mentioned it in letters to his friends only a year or so before his death. As his resentment increased, his reticence became more and more pronounced, and reached such exaggerated proportions that, after his three original communications, he refused to publish anything more on the x-rays; and he also stipulated in his will that all records of his work and all correspondence about the discovery between 1895 and 1900 be burned unopened at his death—a provision, which, unfortunately for the historical record, had to be carried out.

The claims of priority in the discovery arose largely from confusion regarding the reports of previously unexplained accidents caused by the x-rays. Sir William Crookes, whose cathode-ray tubes Röntgen had used in many of his experiments, had observed that unopened boxes of photographic plates were fogged and had complained repeatedly to the manufacturer that they were unsatisfactory. That this effect was caused by x-rays he did not know until their discovery was announced. Others had had similar experiences with photographic plates, but had not investigated the reason for it. A. W. Goodspeed, professor of physics at the University of Pennsylvania in Philadelphia, actually made an accidental x-ray photograph on February 22, 1890, during the process of demonstrating a Crookes' tube. He kept it with a collection of freak photographs, but recalled and unearthed it six years later, when the discovery of x-rays was announced, and proved that it was actually an x-ray photograph. Goodspeed described this in a lecture on the roentgen rays which he gave in February 1896 but added: "We can claim no merit for the discovery, for no discovery was made."

Typical of the inaccurate descriptions and stories about the discovery was the fable of book and key, which was originated by an American student attending Würzburg University and widely circulated in the United States. This gave April 30, 1895, as the date. "Röntgen had a Hittorf tube covered by a light tight paper energized by a coil and was studying the fluorescence of the screen one afternoon, and being called away for a few minutes, he laid the glowing tube upon a book which contained a large flat key, which was being used for a bookmark. A loaded photographic plateholder happened to be lying under the book. When he returned he shut off the current from the tube, took the plateholder with several others and spent the afternoon out of doors, exposing several plates in the practice of his favorite hobby, photography. On developing the plates, he found the shadow of the key bookmark on one of them.

He wondered how this happened and questioned several of the students, but none could explain the incident."

This story has had wide appeal for the general public, and has reappeared in numerous distorted versions of the discovery, but even in its original form, it contains inaccuracies. Laying a glowing tube on a book would necessitate disconnecting the high voltage from the tube and hence no x-rays could be produced. "Studying the fluorescence of the screen" around a Hittorf tube would indicate that the fluorescent effect of the rays had been noticed previously. Neither Röntgen nor any of his intimate colleagues and friends ever spoke of anything connected with the book and key experiment. Röntgen was always most accurate and painstaking in recording experimental data, yet he never referred to any such observation on April 30, 1895. His two senior assistants, one of whom helped to evacuate the Hittorf tubes, did not know about the discovery of the x-rays until Röntgen's announcement in December 1895. If these men who were working rather intimately with Röntgen knew nothing of the discovery, it seems extremely unlikely that a student at the University should have had any inside information as to the work the professor was doing.

One of the most pernicious and persistent rumors was that the discovery was merely an accident and that the first crucial observation of the fluorescence of the screen was made by a laboratory assistant. In his last years, Röntgen once said, "What miserable envious soul must have invented this story."

Of all the criticism and antagonism directed towards Röntgen, the most distressing to him was the attitude of Philipp Lenard, which grew increasingly bitter and puzzling with the years, and continued—so far as Lenard was concerned—after Röntgen's death in 1923.

Of the scientific events antedating the discovery of the x-rays, Lenard's scientific contributions on the properties of the cathode rays immediately preceded and undoubtedly were a principal factor in stimulating Röntgen's researches that led to recognition of the "new" rays. However, Lenard's work and also Röntgen's, rested on the discoveries of many scientific predecessors, especially the experimental work of Hertz—who was Lenard's teacher—and the theories of von Helmholtz. Röntgen gave Lenard due credit for his "wonderful" experiments in his first communication and also mentioned the Lenard vacuum tube as one of those he used in his experimental work. Immediately after the discovery, Lenard's attitude toward Röntgen was friendly, as proved by letters still in existence, but a definite antipathy and bitterness, on Lenard's part, appeared at about the time Röntgen received the first Nobel prize for physics in 1901. From the time when Lenard himself received the Nobel prize in 1905, it seemed clear that he had embarked upon a campaign to minimize the work of Röntgen, and to make him appear a bungler who merely happened accidentally to make a great discovery. He said in his Nobel prize lecture: "In reality I had made several unexplainable observations which I carefully kept for future investigation—unfortunately not started in time—which must have been the effects of traces of wave radiation." In his publications on cathode rays which appeared before Röntgen's discovery, Lenard did not

mention these observations: hence any attempt to give Lenard the credit for discovery of the x-rays is without any historical foundation.

In Lenard's publications during later years, he has either ignored Röntgen or dismissed him and the x-rays (high-frequency rays, as he called them) in a casual footnote. He has tried to perpetrate the idea that anyone who was wide-awake and using a Lenard tube could have discovered the x-rays. He excuses his own failure to discover them by saying that, as a good scientist, he concentrated on his own special line of investigation and postponed the study of strange phenomena until after his original work was completed.

During a controversy in 1929, Lenard intimated that perhaps more data in regard to the discovery of "high-frequency rays" might be revealed at a later date. On August 18, 1929, he wrote to me: "There is no doubt that the road to the discovery led over my researches. At that time I was prevented by external circumstances from pursuing to my satisfaction in every direction the great number of new phenomena that appeared in my studies on cathode rays. But in my opinion, this is not yet the proper time to express myself more thoroughly on the subject than I did in my Nobel prize lecture. That would be only biographical anyway, and what has already been said must suffice for the judicious. With this I believe that I have done everything that the history of science can expect of me on this point at this particular time." Lenard continued later to maintain this enigmatic attitude.

It is interesting that Lenard's extreme nationalism and strong anti-Semitic prejudice assured him an outstanding position in Hitler's regime, and added great weight to whatever he had to say. Hence, with the ascendency of the Nazis, the controversy regarding the credit that Lenard should receive in connection with the discovery of the x-rays assumed distorted and exaggerated proportions that extended outside the realm of a priority claim in the field of physics. In 1935, friends of Lenard made a concerted effort to prove that Röntgen used the Lenard tube in making the discovery, but the evidence was not sufficient to alter the story of the discovery. Just preceding and during the war years, there was considerable discussion, carried out with the help of the Nazi press and some party members, over the position of Röntgen in the annals of science. Articles appeared in certain daily papers suggesting that he had done nothing remarkable, but merely had carried to its inevitable conclusion the work of the great Aryan scientist, Philipp Lenard, Director of the Physical Institute at Heidelberg University.

In 1944, the Physical-Medical Society of Würzburg made application to the Nazi Minister of Post and Telegraph to have a memorial stamp made for Röntgen, marking the fiftieth anniversary of the discovery of the x-rays, similar to the one issued for Robert Koch and other scientists; but the request was rejected with the comment that the proposal was not in order inasmuch as such an honor was reserved only for the illustrious. The Minister of Post, Dr. Ohnesorge, happened to be a good friend of Lenard's. Seven years later, the Ministry of Post of the Federal Republic of Western Germany issued a Röntgen-

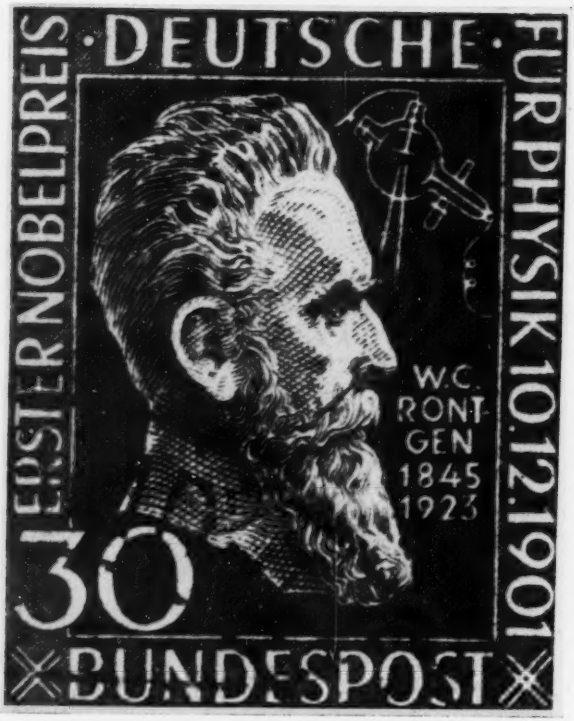

Fig. 1. Röntgen-stamp issued in 1951 by the Ministry of Post of the Federal Republic of Western Germany at the occasion of the fiftieth anniversary of the award of the first Nobel prize in physics to Röntgen.

stamp at the occasion of the fiftieth anniversary of the award of the first Nobel prize in physics to Röntgen (fig. 1).

In Lenard's four volume work, "Deutsche Physik," there is no mention of Röntgen or of Einstein in the text, and the foreword is a diatribe against the Jews. Many persons in Hitler's Germany naturally drew the inference that Röntgen was a Jew. When Lenard was asked directly, "Was Röntgen a Jew?" he replied, "No, but he was a friend of Jews and acted like one."

That Röntgen had good Jewish friends is a fact, for he mentioned them frequently, and with affection, in his letters. It is also true that he condemned anti-Jewish prejudice, as shown in this letter to a friend, written May 12, 1921: "The anti-Semitic incidents in Würzburg of which you write are exceedingly regrettable; it is not much better here (Munich). For instance, there is scarcely an advertisement of rooms for University students which does not contain the statement, 'No Jews,' and I know of one instance when a woman said to a

student who was looking at a room and mentioned his name, which sounded Jewish, 'I do not take any Israelites.' It is a bad sign of the times that decent persons can thus be so rudely insulted."

The record shows that Lenard's animosity towards Röntgen—which would seem to be based on resentment of Röntgen's greater fame—became more intense with the years. The extreme bitterness in his old age (83 years in 1945) was expressed in an interview with Lewis Etter as follows: "I am the mother of the x-rays. Just as a midwife is not responsible for the mechanism of birth, so was Röntgen not responsible for the discovery of the x-rays, which merely fell into his lap. All Röntgen had to do was push a button, since all the groundwork had been prepared by me . . . Without my help the discovery of x-rays would not have been possible even today. Without me the name Röntgen would be unknown. Röntgen was an opportunist who sensed that there was something to be found in experimenting with my tube which he carried out with an eye to fame . . . I was always too modest and did not rush into print. In my letter to Röntgen where I praised him for his great discovery I thought he would reply that he really owed it all to me and my tube, but I waited for this acknowledgement from him in vain."

It is sad and disillusioning that a man like Lenard, whose scientific achievements rank with the best, should be so consumed by jealousy that he should also try to take credit for the great discovery of a fellow scientist. It is also disappointing that one trained in the scientific method should forsake the broad, impartial, honest integrity—the ideal of Truth—of the scientific viewpoint and degrade himself by expressions of narrow partisanship, racial prejudice and petty jealousy and slander. This indicates that some scientists—though fortunately they are few in number—fail to live up to the high ideals of the cause they serve.

It was precisely because he failed to measure up to the high ideal of truth, which is the essence of science, that Lenard found such high favor in the Nazi regime. He tried to add to his own renown by subtracting from Röntgen's glory, and he succeeded temporarily, in his own country, because the environment of the Third Reich was one in which prejudice flourished.

When seen against this distorted background, the character of Röntgen stands out in bold relief. Because he was a man of real integrity—the true scientist in his life as in his work—and hence could not tolerate intolerance, his memory was not revered by the National Socialists. That they could not regard him as illustrious is, in one sense, a measure of his true universality and greatness. In democratic Germany and elsewhere in the world, Röntgen's honor and fame have steadily increased during the almost 60 years that have elapsed since his great discovery.

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THE FRANK E. BUNTS INSTITUTE

Announces the following Postgraduate Continuation Course for September 30 and October 1, 1953.

CURRENT THERAPY IN PEDIATRIC PRACTICE

Tentative Program

Wednesday, September 30, 1953

8:00- 9:00 a.m. . Registration

Morning Session . . . . . R. D. MERCER, M.D., Presiding

9:00- 9:10 a.m. . Welcoming Remarks . . . . . A. C. ERNSTENE, M.D.

9:10- 9:25 a.m. . Rheumatic Heart Disease . . . . . .A. C. ERNSTENE, M.D.

9:25- 9:45 a.m. . Congenital Heart Disease . . . . . .F. M. SONES, JR., M.D.

9:45-10:05 a.m. . Surgery for Congenital Heart Disease . . . . . .D. B. EFFLER, M.D.

10:05-10:25 a.m. Intermission

10:25-10:40 a.m. . Common Problems of Sexual Development .VIOLA STARTZMAN, M.D.

10:40-10:55 a.m. . Research in Poliomyelitis . . . . . .FRED ROBBINS, M.D. (Guest)

10:55-11:40 a.m. . Antibiotic Therapy . . . . . .WARREN WHEELER, M.D. (Guest)

11:40-12:00 a.m. . Question and Answer Period

12:00- 1:30 p.m. . Lunch

Afternoon Session . . . . . .VIOLA STARTZMAN, M.D., Presiding

1:30- 1:45 p.m. . The Diabetic Child . . . . . .E. P. MCCULLAGH, M.D.

1:45- 2.05 p.m. . Abnormalities of Calcium and Phosphorus Metabolism . . . . . .WILLIAM WALLACE, M.D. (Guest)

2:05- 2:25 p.m. . Vomiting in the Newborn Period . . .SAMUEL SPECTOR, M.D. (Guest)

2:25- 2:35 p.m. . Intermission

2:35- 2:50 p.m. . Common Skin Disorders . . . . . .G. H. CURTIS, M.D.

2:50- 3:20 p.m. . Panel Discussion: The Celiac Syndrome . . . . . .RICHARD HODGES, M.D. (Guest)
R. D. MERCER, M.D.
WARREN WHEELER, M.D. (Guest)
SAMUEL SPECTOR, M.D. (Guest)

3:20- 3:40 p.m. . Treatment of Meningitis . . . . . .ROBERT EIBEN, M.D. (Guest)

3:40- 4:15 p.m. . Question and Answer Period

Thursday, October 1, 1953

Morning Session . . . . . .VIOLA STARTZMAN, M.D., Presiding

9:00- 9:05 a.m. . Welcoming Remarks . . . . . .R. S. DINSMORE, M.D.

9:05- 9:15 a.m. . Subdural Hematoma . . . . . .R. D. MERCER, M.D.

9:15- 9:25 a.m.	.	Treatment of Hydrocephalus and Myelomeningocele . . . . . A. T. BUNTS, M.D.
9:25- 9:45 a.m.	.	Mediastinal Masses in Childhood . . . . . D. B. EFFLER, M.D.
9:45-10:00 a.m.	.	Intermission
10:00-10:20 a.m.	.	Plastic Repair of Congenital Defects . . . . ROBIN ANDERSON, M.D.
10:20-10:40 a.m.	.	Diagnosis and Treatment of Masses of the Neck . . . . . GEORGE CRILE, JR., M.D.
10:40-10:50 a.m.	.	Hearing Problems in Children . . . . . H. E. HARRIS, M.D.
10:50-11:30 a.m.	.	Surgical Emergencies in the Newborn . . . . . WILLIAM CLATWORTHY, JR., M.D. (Guest)
11:30-12:00 noon	.	Question and Answer Period
12:00- 1:30 p.m.	.	Lunch
		Afternoon Session . . . . . R. D. MERCER, M.D., Presiding
1:30- 1:45 p.m.	.	Extrophy of the Bladder . . . . . C. C. HIGGINS, M.D.
1:45- 2:00 p.m.	.	Congenital Anomalies of the Genitourinary Tract . . . . . W. J. ENGEL, M.D.
2:00- 2:20 p.m.	.	Proctologic Problems of Childhood . . . . R. B. TURNBULL, JR., M.D.
2:20- 2:30 p.m.	.	Intermission
2:30- 2:50 p.m.	.	Treatment of Minor Foot Abnormalities . . . . J. I. KENDRICK, M.D.
2:50- 3:20 p.m.	.	Panel Discussion: Abdominal Pain in Childhood . . . . . WILLIAM CLATWORTHY, JR., M.D. (Guest) S. O. HOERR, M.D. R. B. TURNBULL, JR., M.D. VIOLA STARTZMAN, M.D.
3:20- 3:40 p.m.	.	Acute Appendicitis . . . . . A. H. ROBNETT, M.D.
3:40- 4:15 p.m.	.	Question and Answer Period

Guest Speakers:

FRED ROBBINS, M.D. — Professor of Pediatrics, City Hospital, Cleveland, Ohio.

WARREN WHEELER, M.D. — Professor of Pediatrics, Columbus Children's Hospital, Columbus, Ohio.

WILLIAM WALLACE, M.D. — Professor of Pediatrics, University Hospital, Cleveland, Ohio.

SAMUEL SPECTOR, M.D. — Professor of Pediatrics, University Hospital, Cleveland, Ohio.

RICHARD HODGES, M.D. — Pediatrician-in-Chief, St. Luke's Hospital, Cleveland, Ohio.

ROBERT EIBEN, M.D. — Assistant Clinical Professor of Pediatrics, Western Reserve University School of Medicine, Cleveland, Ohio.

WILLIAM CLATWORTHY, JR., M.D. — Professor of Pediatric Surgery, Ohio State University Medical School, Columbus, Ohio.

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